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Despite its homology to angiostatin apolipoprotein(a) does not affect angiogenesis.

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Apolipoprotein(a) [apo(a)] contains a kringle domain(IV) homologous to that of angiostatin, a natural angiogenic inhibitor. Because of this structural similarity we suspected that apo(a) could be an inhibitor of angiogenesis. The possible role of apo(a) in microvascular proliferation was studied in an in vivo quantitative model, the disc angiogenesis system (DAS) and compared to angiostatin. Apo(a) and other test compounds were placed in the center of a polyvinyl alcohol foam disc that was implanted subcutaneously in mice. After 14 days, the disc was removed and vascular growth into the disc was measured. Apo(a) did not affect spontaneous vessel growth into the disc, while angiostatin suppressed this growth and basic fibroblast growth factor (bFGF) increased it. Additionally, apo(a) did not modify the vascular growth induced by bFGF. Transgenic mice expressing the human apo(a) gene were used to study the systemic effect of apo(a): neither an increase nor a decrease in vascular growth was detected. Our results suggest that apo(a) is unlikely to play a significant role in the control of angiogenesis. Furthermore, our experiments confirm the inhibitory effect of angiostatin not only on induced angiogenesis but also on baseline, spontaneous angiogenesis. Copyright 1998 Academic Press.

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